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THE SHORT-TERM EFFECTS OF LEAD ON DOMESTIC AND WILD ANIMALS

by

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FOREWORD

Effective regulatory and enforcement actions by the Environmental Protection Agency would be virtually impossible without sound scientific data on pollutants and their impact on environmental stability and human health. Responsibility for building this data base has been assigned to EPA's Office of Research and Development and its 15 major field installations, one of which is the Corvallis Environmental Research Laboratory (CERL).

The primary mission of the Corvallis Laboratory is research on the effects of environmental pollutants on terrestrial, freshwater, and marine ecosystems; the behavior, effects and control of pollutants in lake systems; and the development of predictive models on the movement of pollutants in the biosphere.

This report presents a discussion on the extent of lead found in wild and domestic animals and some of the short term effects of this toxic substance.

A.F. Bartsch
Director, CERL

ABSTRACT

Small quantities of lead, a ubiquitous and toxic element, may be found in practically all species of plants and animals. The list of animals, both domestic and wild, reportedly intoxicated by lead is impressive.

The sources of lead poisoning vary with species of animals. Lead base paints, used motor oils, spent lead shot and pastures contaminated by lead smelters seemingly have been most often incriminated.

The lesions associated with lead intoxication may vary widely both within and between species of animals. Lesions and symptoms of the central nervous system are the most prominent.

Toxic levels for various species as reported in the literature vary widely and seemingly a single toxic dose for each species, as yet, has not been definitely established.

The diagnosis and treatment of lead intoxication may become laborious and time consuming. Most symptoms reported involve central nervous system dearrangement. Treatment of most clinical cases is disappointing generally because of the acute nature of lead poisoning.

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Section 1

CONCLUSIONS

From data reported in the literature crude estimates of the occurrence of lead poisoning may be derived. Approximately 160,000 cattle are annually exposed to toxic levels of lead and 20,000 acute cases occur per year. Of these, 15,000 animals succumb to lead poisoning. In addition, it is estimated that 1,000,000 migratory waterfowl succumb to lead poisoning due to consuming spent lead shot.

The metabolism of lead compounds is very complicated and variable both within and between species. For most mammals including man 1-6% of the lead taken orally will be absorbed. One may estimate for ruminants that 83.6% is excreted through the feces; 14.8% through the urine, and 1.6% through the milk. Lead, once taken internally is retained by the body; particularly, the bones tenaciously retain lead and may account for as much as 80% of the total body burden.

The exact mechanisms giving rise to the pathological lesions associated with lead poisoning have not been clarified. This is particularly true for chronic encephalopathy and nephropathy. Several enzymes are known to be inhibited by lead and among this group are several enzymes shown or suspected of being involved in renal tubular transport.

Even with the voluminous literature concerning lead, toxic levels for various species of animals have not been precisely determined, nor is there general agreement on what constitutes a safe level of intake for extended periods of time.

Generally, the treatment of lead poisoning is disappointing because of the acute nature of most clinical cases. Thus more emphasis should be placed on preventive rather than therapeutic programs to avoid the losses due to lead poisoning.

Section II

INTRODUCTION

Lead is one of the oldest metals used by mankind; Romans and medieval Britons constructed lead water pipes (1). Usage has expanded until today the total annual U.S. consumption in 1968 was 1,328,970 tons (2). Approximately 184,316 tons annually are emitted to the atmosphere from sources such as combustion of gasoline, fuel oil, and coal in addition to several other industrial sources. The most abundant lead ore is Galena (PbS) (1). Approximately 32 percent of the U.S. consumption is obtained from primary smelters, 38 percent from secondary recovery, and 30 percent from imports (2).

Lead is an ubiquitous and toxic element. Small quantities can be found in practically all living tissues of plants and animals. Soils, limestone, sandstone, shale and igneous rocks contain quantities varying from 7 to 20 ppm (3). Much has been written about lead, and thousands of cases of lead poisoning have been reported in both the human and veterinary medical literature. The species of animals that have either died or been obviously ill from increased lead intake include:

A. Farm livestock and pets

1. Horses
2. Cattle
3. Swine
4. Dogs
5. Sheep (this review failed to find any reports involving naturally occuring lead poisoning).

B. Wild animals

1. Ducks
2. Geese
3. Andean Condor
4. Doves
5. Quail
6. Pheasants
7. Falcon

C. Zoo animals

1. Non-human primates
2. Leopards
3. Australian fruit bats
4. Parrots
5. Polar bear
6. Lorikeets
7. Ferrets

Most of the reported incidents of poisoning involved small numbers of animals. However, contamination of vegetation by industrial operations(4-13) often results in death to several animals at one time. Because lead is the most common accidental poison of farm animals (14), it is of considerable economic importance to agriculture.

Section III

SOURCE OF LEAD AND THE EPIZOOTIOLOGY AND LEAD TOXICOSIS

The diagnosis of lead poisoning is not precise in all cases. For this reason, knowledge of the epizootiology of lead poisoning can help in arriving at a definitive diagnosis, in addition to determining the occurrence of the disease and defining the circumstances of exposure.

EPIZOOTIOLOGY

Although definite estimates of the incidence, morbidity and mortality in domestic and wild animals is lacking, the literature contains some information concerning the epizootiology of lead poisoning. Reports often state that the morbidity is low and the mortality or case fatality rate is high (14, 15). Lead poisoning is diagnosed more frequently in cattle and dogs than other species (14).

For cattle, the incidence of lead poisoning has been estimated as approximately 167 cases per year per 1,000,000 animals (2). However, several tenuous assumptions were made in order to derive this estimate. The proportional mortality for lead poisoning (e.g., no. of animals dying of lead poisoning per all animal deaths) in cattle is estimated to be 1.7 percent for calves and 4.5 percent for older cattle (16). The morbidity of animals exposured to toxic levels of lead is estimated to be 11-15 percent and the case fatality rate is 55-85 percent (17). Two possible explanations are offered to explain the above estimates. Either, few animals--only the most serious cases--are presented to diagnostic laboratories or few exposed animals receive doses of sufficient magnitude to cause illness. If one can accept the incidence of lead poisoning to be approximately 0.016 percent (2) per year and the morbidity of animals exposed to toxic lead levels to be 11-15 percent (17), then one may conclude that the ratio of exposed to unexposed cattle is 1:600 to 1:900. With a total 1969 cattle population of 123,748,000 in the U.S., one can estimate that between 137,000 and 190,000 cattle are annually exposed to toxic levels of lead and that 20,000 cases of lead poisoning occur per year.

Lead intoxication has a definite seasonal trend for cattle (17), dogs(19) and human beings (2). During a five year period (1965-1970), 41 percent of the bovine cases of a large diagnostic center occurred during April, May and June (17). In contrast, lead poisoning in dogs more often is diagnosed in summer and fall (19). The seasonal trend for cattle, at least in Iowa, coincides with the time when cattle are released to spring pastures (17).

Very little information is available on the age distribution of lead poisoning. For dogs, poisoning apparently occurs more frequently in younger animals, possibly because of their indiscriminate chewing habits (2, 19).

The incidence of lead poisoning is unknown for practically all species of wild animals. However, an estimated one million ducks, geese and swans die annually in the U.S. of lead poisoning (20).

The sources of lead poisoning are varied and often depend on such factors as the characteristics of the animal and its peculiar husbandry considerations. Consumption of spent lead shot for wildlife species such as ducks, geese, swans, mourning doves, pheasants, condor and falcons most often has been incriminated (21-25) as the source of lead poisoning. The consumption of flaking-paint containing lead is a common source for zoo animals (26-28). For domestic and pet animals, the sources of lead poisoning are more variable. In one study (17) the sources of lead incriminated for cattle were paint (29 percent), oil (25 percent), unknown (24 percent) junkpiles (11 percent) grease (6 percent) and batteries (5 percent). Table 1 lists the most often quoted sources of lead by species (4,5,6,7,8,9,10,17, 29-33).

Table 1. Sources of Lead Poisoning for Various Species of Livestock

Species	Sources
Cattle	Lead base paint, used motor oil, grease, used batteries, junkpiles containing lead, putty, roofing material, linoleum contaminated water, contaminated feed contaminated pastures from lead smelters
Horses	Contaminated pastures from lead smelters
Swine	Lead base paint
Dog	Paint, linoleum, shot gun slugs, curtain weights, plaster

Some of these diverse sources are more potent in terms of their lead content. Grease may contain as much as 450,000 ppm (17) and oil as much as 600 ppm (34). Horses and cattle have died grazing on contaminated pastures that contained as much as 2,000 ppm (35) of lead.

The epizootiology of lead poisoning may be briefly summarized:

1. For Farm Animals

   a. The disease occurs more often in spring.
   b. The morbidity is low and mortality high.
   c. The sources most often incriminated include paint, used oil, grease, and discarded batteries.

2. For Dogs

   a. The disease occurs more often in summer and fall.
   b. Most often it is a disease of young dogs.
   c. Several sources have been suggested.

3. For Wildlife

   a. Consumption of spent lead shot is the primary source.
   b. Approximately 1,000,000 migratory birds are lost annually.

4. For Zoo Animals

   a. Lead base paint consumed after it has flaked from cages and enclosures is the primary source.

Obviously, the salient features of a program to reduce the occurrence of lead poisoning would involve:

1. On the farm, implement clean husbandry practices. In particular

   a. Don't drain the oil from motor vehicles in pastures.
   b. Don't discard batteries, paint buckets, or used grease cans in pastures.
   c. Erect fences to keep animals out of old junk piles

2. With non-lead paint, thoroughly clean and repaint all cages for animals.

3. For wildlife, a preventive program (being implemented)(2) involves developing a substitute for lead shot.

Section IV

METABOLISM OF LEAD

ABSORPTION

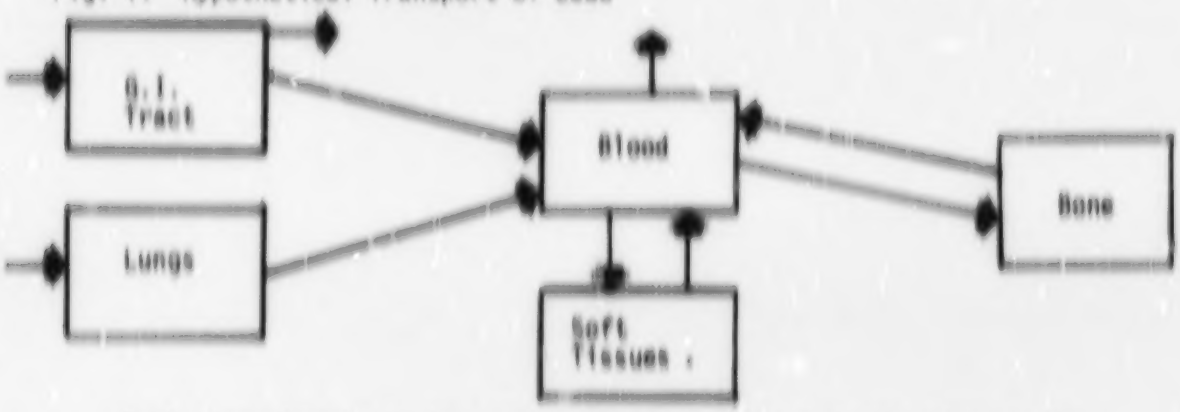
The absorption of lead compounds is quite complex and involves three different routes: inhalation, ingestion, and absorption through the skin. Of these inhalation is most efficient, and skin absorption is the least efficient. For man, 14-45 percent of inhaled lead is retained, whereas only 5-15 percent of that ingested is retained (36). Factors that may modify retention of lead particulates in the lungs are the particle size, mucous flow, ciliary action, and the alveolar clearance rate (36).

The percent retained by ingestion or the digestive coefficient is computed by determining the ratio--the portion retained/portion consumed. The digestive coefficient of lead varies widely between species from 5-15 percent in man (36) to as little as 1-3 percent in cattle and one percent in rabbits (37). For many toxic metals, the rate of absorption depends on such factors as the chemical form of the lead, the age of the animal, the pH of the gastro-intestinal tract, the gut transient time, the gut microflora, and the presence or absence of other metals. For example reducing the calcium level in the diet may increase the lead absorption in rats (38). In contrast vitamin D has a reverse effect (39). Furthermore lead absorption by the gastro-intestinal tract is greater in young than in old rats (40,41).

TRANSPORT, DISTRIBUTION AND EXCRETION

Once lead is absorbed, it enters into a state of exchange between tissues and the blood. The various organs retain different amounts of lead. Transport, distribution and excretion of lead may be best conceptualized as a series of compartments interconnected with one another via the blood (Figure 1).

Fig. 1: Hypothetical Transport of Lead



Each compartment receives and will retain lead from the blood. The retention time of the lead depends on its biological half-time $t_{1/2}$, which in turn depends upon the strength of the bond between lead and the binding tissue.

If the transfer from a tissue can be assumed to be a first-order decay, the $t_{1/2}$ is the time required for 1/2 of the present burden of lead to be transferred from the tissue to the blood. In many biological systems, the transfer of material between compartments can be simulated by assuming a first order decay (42,43). Then the transfer rate depends only on the concentration in the originating compartment and the transfer coefficient for that compartment. The transfer coefficient, k, is related to $t_{1/2}$ by the equation:

$$k = \frac{\ln(2)}{t_{1/2}}$$

One can appreciate that the concentrations in the various organs will change with time after a single dose of lead is absorbed and that these concentrations will depend partly upon the $t_{1/2}$.

Since blood is the medium that transports lead, it assumes an important role in the metabolism of lead. Based on tracer studies (44) in the dog and wet chemical analysis of tissues and blood from sheep (45), one may conclude that blood contains more than one compartment. Results reported in dogs (44) indicate at least three compartments. Some suggest (36) four compartments: red blood cells (RBCs), specific metalloproteins, plasma proteins and low molecular weight plasma proteins. The transfer rate is much slower (that is the $t_{1/2}$ is longer) for RBCs than for plasma or sera; thus, for the most part, the concentration would be greater in the RBCs than in the plasma. Forty and one-half hours after receiving an oral dose of lead, the RBC to plasma ratio was 122 in a calf (46). In contrast, this ratio was estimated to be 11 in sheep, 40 hours after an intravenous dose (45). This latter

data (46) show that the $t_{1/2}$ is longer for RBCs than for serum, and consequently, the fraction bound to serum would be transferred more readily to other tissues. Several factors that combine to maintain the level of lead in the blood include the initial dose of lead, the $t_{1/2}$ of the compartments in the blood, and the $t_{1/2}$ of the various compartments perfused by the blood. From the results reported in dogs (44) after 20 days the level in the blood fell to an estimated 5.8 percent of the level at the time of injection and, after an additional 260 days, further fell to approximately 2.5 percent. This fact demonstrates the persistence of lead once it is taken internally.

The distribution of lead in the various tissues at any one time after a dose of lead depends on the perfusion of the tissue, the fraction in the blood that is diffusable, and the $t_{1/2}$ of the perfused tissues. Concentration of lead in different tissues demonstrate remarkable variations. Generally, lead is concentrated in bone, liver and kidney tissues--concentrations as much as 100 times greater than in plasma or muscle. In a calf analyzed 42.5 hours after injection with lead, 34 percent of the total body burden was found in the liver, 33 percent in the bone, and 21 percent in the blood (46). In rabbits 96 hours after injection, 21 percent of the lead was found in the liver, 70 percent in the bone, and one percent in the blood (45). The kidney concentrates lead but, because of the relative weight, contains appreciably less of the total body burden. A publication reported data for a calf that consumed a daily average of 0.1 mg of lead for 169 days (47). If one assumes total body ratios of .04 for blood, .04 for bone, and .03 for liver, one can estimate that approximately 80% of the total body burden is located in bone and 5 percent in the liver. At low intake levels and after a sufficient length of time, an equilibrium is reached where excretion equals intake. Then the concentrations in the various compartments do not change, and that compartments (bone for lead) with the longest $t_{1/2}$ will contain an appreciable amount of the total body burden. The $t_{1/2}$ for bone in dogs was estimated to be 7600 days (48). In contrast a $t_{1/2}$ of blood was estimated to be 3.5 days(42).

Lead absorbed by the gastrointestinal tract can be excreted via four routes: (1) glandular excretion and epithelial shedding of the gastrointestinal tract, (2) biliar excretion, (3) urinary excretion, and (4) mammary glandular excretion (36).

The relative importance of these routes is unknown for many animals. After intravenous injection of lead, the ratio of the fecal (i.e., the sum of the glandular excretion and epithelial shedding) to total excretion can be computed. In the dog this ratio is approximately 0.65 (44); in sheep over a 10 day collection period the ratio was 0.84 (36). The discrepancies between this ratio for dogs and sheep may be due to the fact that the dose of lead received by the dog (44) was much less on a per weight basis than that received by the sheep (37) or due to differences in the metabolism of lead. Toxic doses of lead may change excretory functions and result in changes of excretory pathways. For example, when cadmium causes renal damage, cadmium excretion increases (36).

Mammary gland excretion is much less than urinary excretion. From data collected 6 days in one cow (46), the mammary gland excretion was 10 percent of the value for urinary excretion.

Combining the data from sheep (37) and cow (46), one can compute a crude excretion ratio: fecal excretion--83.6 percent; urinary excretion--14.8 percent; and mammary excretion--1.6 percent.

Section V

LESIONS ASSOCIATED WITH LEAD INTOXICATION

The functional and pathological effects quoted in the literature are quite irregular and variable for man and various species of animals.

ANEMIA

When the synthesis of RBCs and their components is decreased anemia occurs. In most cases of lead intoxication, anemia is not a predominating clinical sign; however, the toxic effects of lead have motivated research to understand porphyrin metabolism which is controlled by several enzymes (49). The list of enzymes that lead inhibits or enhances is impressive. Many of these effects are demonstrable in vitro only, and in some instances, at concentrations that never have been observed in clinically ill individuals (49).

Heme synthesis may be briefly outlined as (2):

$$\text{Succinyl CoA} + \text{Glycine} \xrightarrow{\text{ALA synthetase (ALAS)}} \text{δ-amino levulinic acid (ALA)} \quad (1)$$

$$\text{ALA} \xrightarrow{\text{ALA dehydrase}} \text{porphobilinogen (PBG)} \quad (2)$$

$$\text{PBG} \longrightarrow \text{uroporphybinogen III (URO III)} \quad (3)$$

$$\text{URO III} \xrightarrow{\text{urogenase}} \text{coproporphybilinogen III (copro III)} \quad (4)$$

$$\text{Copro III} \xrightarrow{\text{coprogenase}} \text{protoporphyrin IX (proto IX)} \quad (5)$$

$$\text{Proto IX} \xrightarrow{\text{Heme synthetase}} \text{Heme} \quad (6)$$

Steps (1), (2), (3 or 4), (5) and (6) have been demonstrated to be inhibited by lead (2, 49). Overt clinical lead poisoning is accompanied by increased ALA and coproporphyrin in the urine and blood (50). In vitro studies suggest that steps (2) and (5) are most sensitive to lead inhibition (47). Depressed ALA dehydrase activity in erythrocytes and

elevated urine and serum ALA have been observed in cattle chronically poisoned (51). In addition to inhibiting heme synthesis lead apparently suppresses globulin synthesis (49).

Other anemia-related abnormalities that have been observed in man and animals include:

1. Shortened survival of RBCs (52)
2. Reticulocytosis (2,33,53)
3. Decreased hemoglobin (53, 54, 55)
4. Basophilic stippling of RBCs (2)
5. Siderocytosis (54)
6. Increased osmotic and mechanical fragility (2,49)
7. Loss of intracellular K+ ions (49)
8. Immature forms of RBCs in general circulation (33)
9. Erythroid hyperplasia of the bone marrow (54)
10. Mitosis of erythrocytes in the peripheral blood in fowl (22,56).

Basophilic stippling, often mentioned as a symptom of lead poisoning, is due to accumulations of cytoplasm and nuclear fragments along with non-heme iron in developing erythrocytes (2). In general, the anemia associated with lead poisoning is not severe and is difficult to distinguish from anemia due to other causes.

NEPHROPATHY

In man, renal insufficiency is a late sequela of lead poisoning (1,2,54). The pathogenesis of renal insufficiency is not clearly defined (2) and apparently occurs in those individuals who are exposed to lead over long periods of time (1,2,54). Lead-induced renal insufficiency is characterized by glycosuria and hyper-amino aciduria (2, 54). In addition to amino aciduria and glycosuria extreme exposure can cause hypophosphatemia and hyperphosphaturia (Fanconi syndrome) (2). Uric acid excretion sometimes is reduced causing saturine gout (2).

Most clinical findings point to renal tubular dysfunction. Electron microscopic and biochemical studies demonstrate alterations in mitochondrial structures and cellular oxidation processes (58,59). The enzymes, alkaline phosphatase, carbonic anhydrase, ATPases, and cytochrome oxidase all inhibited by lead (49)--are known or suspected of being involved in renal tubular transport mechanisms (60). The most consistent lesion associated with lead poisoning is intranuclear inclusion bodies of the proximal tubules. These inclusions, believed to be lead protein complex (61), are reported in various species of mammals and birds dying of lead poisoning (22,56,62,63,64). Although cloudy swelling and degenerative changes of the tubules can be observed, those conditions are usually mild and inconspicuous (15,65).

In summary, the significance of renal insufficiency in lead poisoning of domestic animals is unknown and has not been described, probably because domestic animals are not exposed to lead long enough for renal insufficiency to be observed.

NERVOUS SYSTEM

Symptoms of central nervous system dearrangement predominate in most diagnosed cases of lead poisoning. In man acute encephalopathy due to lead intoxication is characterized by rapid onset of seizures, often followed by coma. In acute encephalopathy (2) vascular injury is the lesion most often associated with edema of the brain. In contrast, patients with chronic encephalopathy develop progressive mental deterioration with loss of motor control (2). The etiology of chronic encephalopathy is not clear and may be due to several other factors.

Encephalopathy dominates in most diagnosed cases of lead poisoning in domestic animals. Apparently, as in man, vascular injury is the initiating lesion (55). In cattle, brain lesions progress from a prominence of endothelial cells of the capillaries to the development of edema foci and neuronal degenerative changes. These focal lesions develop a laminar pattern accompanied by enlargement of astrocytes. Chronically ill animals develop lesions in the tips of the gyri, accompanied by capillary endothelial proliferation, astrocytic proliferation, spongy degeneration, neuronal necrosis, cavitation due to loss of cells, and capillary proliferation in these areas.

In dogs, the reported lesions are similar to those in cattle (66). Damage to the small vessels include necrotic endothelium, usually associated with laminar necrosis in the cerebral cortex. Gliosis and capillary proliferation are observed in the chronically ill. In per acute cases, significant lesions may not be observed (67).

Lesions of the central nervous system are not severe in birds. Chickens tolerate relatively high doses of lead (68), and CNS lesions are not well defined in the literature. Geese experience cerebral edema with serious fluid accumulation in the spaces of Virchow-Rubin (69). However, routine staining procedures failed to demonstrate any significant lesions in poisoned ducks (22).

PERIPHERAL NEUROPATHY

In man, peripheral neuropathy is observed and characterized by motor involvement with no sensory nerve aberrations. Chronically poisoned experimental animals develop segmental demyelination of some nerve fibers (2). Electrophysiological studies of poisoned baboons revealed no abnormalities of nerve conduction (70). Horses with lead poisoning exhibit signs of severe inspiratory dyspnea (4), possibly having the same histological basis as peripheral neuropathy observed in man (15).

Section VI

OTHER FUNCTIONAL EFFECTS ASSOCIATED WITH LEAD POISIONING

Numerous organ-systems reportedly (2) impaired by lead intoxication include:

1. Decreased iodine uptake
2. Decreased gonadotrophic hormones
3. Abnormal function of the pituitary-adrenal axis
4. Myopathy of skeletal muscles
5. Cardiac arrhythmias
6. Decreased fertility
7. Abnormal serum albumins and serum globulin levels (54)
8. Elevated serum cholesterol levels (54)

As in man, a variety of abnormalities have been reported in domestic and experimental animals. Apparently lead is immuno-suppressive evidenced by reduced lysozyme in dogs poisoned with lead (54). Lead decreases the resistance of mice to Salmonella typhimurium (71), in addition to decreasing the number of splenic cells producing both IgM and IgG antibodies (72). Lead poisoning in the dog produces acidophilic intranuclear inclusion bodies in the liver. Liver function tests reported in dogs (73) and cattle (55) occasionally indicate elevated levels of some enzymes (e.g., serum glutamic oxalacetic transaminase, serum pyruvic transaminase, and ornithine and carbamyl transferase).

The gastroenteritis associated with lead intoxication varies from no lesions in cattle (15) to severe lesions in sheep (29). One dog experienced a perforated gastric ulcer with lead poisoning (74). In another report, 24 percent of poisoned cattle had some form of gastroenteritis (17).

Abnormal changes in bony tissue have been reported in horses suffering from lead poisoning (63). Their lesions included edematous synovial membranes, erosion of joint surfaces, and exostosis. Radiographs of experimental poisoned dogs demonstrated radio-opaque lines of the distal radii and thoracic spines (73). One study of the interactions of lead, zinc, cadmium in pigs(75) reported radio-opaque lines in the metaphysis. Other changes associated with lead poisoning include chromosome damage (76), abortion (76,77), reduced fertility (76), and endocardial and epicardial hemorrhages (16).

The relative frequency of the lesions and reported functional effects are unknown and probably vary widely even within species. One report (17)

lists the frequency of gross lesions in cattle as: gastroenteritis (24 percent), petechiation of the epicardium and myocardium (21 percent), pulmonary congestion (16 percent), and degenerative changes of the kidneys (16 percent). Other lesions with reported frequencies of less than 10 percent are: fatty liver, pale muscles, petechiation of subcutaneous tissue, thymus, and trachea; cystitis; opaque cornea; ocular hemorrhage; edema and hyperemia of the brain; and swollen mesenteric lymph nodes.

The lesions reported in ducks and geese poisoned with lead contrast sharply with that reported in mammals. In ducks the most notable lesions are atrophic pectoral muscles and emaciation with loss of fat deposits (22). Other lesions include impaction of the proventriculus, small spleen, and vacuolization of hepatic cord cells. In addition to the lesions reported in ducks, geese, experience marked cephalic edema involving eyelids, submandibular region, and the brain (69).

Section VII

TOXIC LEVELS OF LEAD FOR VARIOUS SPECIES OF ANIMALS

The toxic and/or lethal levels of lead, listed in Table 2 by species of animals, reported in the literature vary widely.

Table 2

TOXIC LEVELS OF LEAD (ORAL DOSE)

Species	Reference	Comments
Cattle	29	Single lethal dose for calves 200-400 mg/kg B.W.
	29	Single lethal dose for adult cattle 600-800 mg/kg B.W.
	29	Adult cattle will tolerate 6 mg/kg B.W./day for 2-3 years
	29	Adult cattle will tolerate 250 ppm of lead in ration for 2-3 years
	4	6mg/kg/day (equivalent to 275 ppm in the ration) is minimal chronic fatal dose.
	35	5 out of 8 animals died grazing pasture containing a median of 516 ppm of lead
	6	5 out of 15 animals died and an additional 5 were symptomatic grazing pasture containing 350 ppm.
	61	1 out of 6 cows died after consuming 5 mg/kg B.W./day for 69 days.
Horses	35	2 out of 2 horses died grazing pastures with median concentration of 516 ppm.

	4	2.4 mg/kg/day equivalent to 150 ppm in ration is the minimum chronic fatal dose (for 6 months)
	78	4 out of 9 animals died and an additional 2 were symptomatic when grazing pastures for 4 months containing 200-840 ppm.
	66	5 out of 6 young horses and 1 out of 22 older animals were symptomatic when grazing pastures containing 38, 90 and 246 ppm.
Sheep	79	2 out of 2 sheep apparently normal for 45 days consuming 5 mg/kg/day.
	79	2 out of 2 pregnant animals aborted and died on 59th and 106th day consuming 9 mg/kg/B.W./day
	29	1 out of 1 pregnant sheep aborted when consuming 1 mg/kg B.W./day for 50 days.
	29	1 out of 1 died when consuming 8 mg/kg B.W./day for 220 days.
	76	10 out of 10 were asymptomatic for 26 weeks consuming 4.5 mg/kg b.W./day
	82	4 out of 4 were symptomatic when consuming 100 mg/kg B.W./day for 30 days.
Dog	67	3 mg/kg B.W./day for 180 days produces symptoms
	67	10-30 mg/kg B.W./day produces symptoms from 13th day
	67	600 mg/kg/day for 2 days is lethal
	73	Consuming 100 ppm in the ration produces symptoms
Poultry	80	100 ppm of PbAC in ration for 4 weeks asymptomatic
	80	1000 ppm in the ration for 4 weeks depressed weight gains
	56	2000 ppm in the ration for 3 weeks asymptomatic

	56	5000 ppm in ration for 3 weeks produced necrosis of renal tubules
	65	Feeding 160 mg/kg/day for 35 days asymptomatic
Ducks	22	10 out of 10 developed symptoms after consuming eight #6 lead shot.
	63	Consuming 6 mg/kg B.W./day for 137 days was asymptomatic
	83	Consuming 6-12 mg/kg B.W./day had average survival times of 28 and 25 days.
Fish	19	Toxic or lethal levels vary widely depending on the kind of water and length of exposure. Vary from 0.01 to 63 ppm.
Other aquatic life	19	Toxic levels for daphnia and cyclops varied from 0.01 to 120 ppm.

The fatal or minimal toxic doses reported in the literature are difficult to interpret. From Table 2, assuming a safety factor of 2-3, older cattle should tolerate 2 mg/kg B.W./day or 80-100 ppm in the ration for 2 to 3 years. Sheep should tolerate 10 mg/kg B.W./day or, assuming that sheep consume approximately 3 percent of their body weight per day, they should tolerate 50 ppm of their ration for approximately one-half year. However, pregnant sheep have aborted at these levels (29). Assuming a horse consumes 2.1 percent of its body weight per day, 1.0 to 1.2 mg/kg B.W./day or 40-50 ppm in the ration for time periods of less than one-half year should be tolerated. These implied safe levels were computed by making several assumptions; consequently, less subtle effects could be observed at levels of lead intake lower than these.

Section VIII

SIGNS, SYMPTOMS AND DIAGNOSIS

The diagnosis of lead poisoning depends on identifying these factors (14):

1. Opportunity for exposure and the sources.
2. Symptoms compatible with lead poisoning.
3. Concentrations of lead in fluids, tissues, and organs of intoxicated animals and concentrations of lead in the source.
4. Gross and microscopic lesions compatible with lead poisoning.

In some extreme instances, test animals may have to be exposed to the source to determine effects of that particular lead concentration.

SIGNS AND SYMPTOMS

The gross symptoms observed in lead poisoning of domestic animals result from disturbances of the gastrointestinal tract and central nervous system. Most cases of intoxication are acute or subacute; consequently, the course of the disease will be short, about 2-3 days (14).

Cattle

Experimental evidence collected in young calves indicates that 2-3 days after exposure symptoms will be observed; however, this time interval is modified by initial dose (24). In fatal cases, the first symptoms observed may be impressive; uncontrolled vocalization, extreme ataxia, nystagmus, excessive salivation, sudden collapse, and death (29). Generally lack of appetite with diarrhea and constipation will be observed. In some instances marked anemia is observed.

The frequency of some symptoms for cattle have been reported as (17):

1.	Blindness	51%
2.	Excessive salivation	46%
3.	Muscular twitching	33%
4.	Hyperirritability	33%
5.	Convulsion	32%
6.	Depression	32%
7.	Grinding teeth	24%
8.	Ataxia	18%

9.	Circling	16%
10.	Bellowing	13%
11.	Diarrhea	10%
12.	Pushing against objects	10%

SHEEP

Generally, the symptoms of sheep contrast with those seen in cattle; rather than excitement, depression often is reported (30). Progressive anorexia, in addition to constipation and diarrhea (29), lethargy and ataxia are typical symptoms.

HORSES

Inspiratory dyspnea, "roaring" after exercise, is the most often reported symptom of horses (4,84). Pharyngeal paralysis with regurgitation also occurs, and severe cases evidence ataxia, paralysis, and clonic convulsions (84).

DOGS

Symptoms for dogs are similar to those reported for cattle including excessive salivation, uncontrolled jaw movements, hyperesthesia of the skin and recumbency with pedal movements (67). Onset may be very rapid with apparent recovery from an acute episode following by vomiting (67). Pharyngeal paralysis also is reported (30).

DUCKS AND GEESE

Severe weight loss, marked weakness, and atrophy of pectoral muscles are usually reported in ducks and geese (22,69). Cephalic edema and characteristic high pitched vocalizations in geese, contrast with that reported in ducks (69).

FISH

Fish residing in fresh water containing lead ions develop a coagulated film of mucous over the entire body, particularly noticeable over the gills (2). This condition frequently occurs in fish exposed to other heavy metals.

Section IX

CLINICAL PATHOLOGY AND CHEMICAL ANALYSIS

Many of the clinical pathologic observations noted with lead poisoning are associated with the development of anemia. Cattle experience hemoglobin levels of 5 gm/100 ml and packed cell volumes of 20 percent(55) as well as elevated reticulocyte counts (19,55). Serum glutamic oxalacetic transaminase and serum glutamic pyruvic transaminase are elevated (73); however, in some instances these enzymes are not elevated (55). Increased prophyrins in the urine seemingly is an inconsistent finding (53). Delta-amino levulinic acid (D.A.L.A.) in the serum and urine of man becomes elevated before the appearance of symptoms (2). In cattle(49) and sheep(85) elevated D.A.L.A. has been reported. Stippling of red blood cells often is reported; however, conditions other than lead intoxication cause stippling of dog RBCs (86). A count of 15 or more stippled cells per 10,000 may suggest lead poisoning. Other findings reported in intoxicated animals include: lead lines in the metaphyses and epiphyses of the radii (33,65,73) and epithelial casts in urine sediment (19).

Chemical analysis of fluids, tissues, and sources is the most frequently used method to support the clinical diagnosis of lead poisoning. Kidney, liver, and blood tissues often are analyzed for lead content. Ten, 20, and 0.35 ppm in the liver, kidney, and blood respectively are levels indicative of lead poisoning (4,30). Others suggest 50 ppm in the kidney cortex and 10 ppm in the liver as significant (29). Levels considered significant in the dog are: blood (35 µg/100 ml); urine (75 µg/liter); hair (80 µg/gram); and liver (3.6 µg/gram)(87).

The antemortem diagnosis of lead poisoning in domestic animals probably is not as crucial as for human beings. Primarily because the course of the disease is more acute and a few animals will have succumbed before the problem becomes evident. The differential diagnosis should include any disease of the central nervous and gastrointestinal system (17).

Section X

TREATMENT

Because of the acute nature in most domestic animals, the treatment generally is disappointing. The treatment regimen may include (85):

1. Sedation for convulsive seizures,
2. Small oral doses of magnesium sulphate,
3. Parenteral or forced feeding
4. Ethylenediaminetetracetate (EDTA).

The recommended dosage of EDTA varies. Some recommend a 12.5 percent solution given at the rate of 1.0 g/30 lb (13.6 kg/day) (84). The dose should be divided in two doses given intravenously and very slowly. The dose can be repeated until symptoms improve. Others suggest a 10 percent solution given intravenous at 1.0 gm/30 lb (13.6 kg)/day limiting the total dose to 5 gm/30 lb (13.6kg) per week suspended for one week after 7.5 gm/30 lb(13.6kg) has been given. Toxic signs include accelerated heart and respiratory rates with muscular tremors (94).

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16. ABSTRACT

Small quantities of lead, a ubiquitous and toxic element, may be found in practically all species of plants and animals. The list of animals, both domestic and wild, reportedly intoxicated by lead is impressive.

The sources of lead poisoning vary with species of animals. Lead base paints, used motor oils, spend lead shot and pastures contaminated by lead smelters seemingly have been most often incriminated. The lesions associated with lead intoxication may vary widely both within and between species of animals. Lesions and symptoms of the central nervous system are the most prominent. Toxic levels for various species as reported in the literature vary widely and seemingly a single toxic dose for each species, as yet, has not been definitely established.

The diagnosis and treatment of lead intoxication may become laborious and time consuming. Most symptoms, reported involve central nervous system dearrangement. Treatment of most clinical cases is disappointing generally because of the acute nature of lead poisoning.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
animal diseases lead poisoning animal nutrition veterinary medicine	lead poisoning in wild and domestic animals sources and effects of lead poisoning in animals	02/E 06/F,T

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